L7 ANSWER 134 OF 137 MEDLINE on STN DUPLICATE 67
ACCESSION NUMBER: 88317012 MEDLINE
DOCUMENT NUMBER: 88317012 PubMed ID: 3166071
TITLE: Cytogenetic analysis of melanocytes from premalignant **nevi** and melanomas.
AUTHOR: Cowan J M; Halaban R; Francke U
CORPORATE SOURCE: Department of Human Genetics, Yale University School of Medicine, New Haven, CT 06510.
CONTRACT NUMBER: CA-04679 (NCI)
CP-21037 (NCI)
GM-26105 (NIGMS)
SOURCE: JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1988 Sep 21) 80 (14) 1159-64.
Journal code: 7503089. ISSN: 0027-8874.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198810
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 20000303
Entered Medline: 19881013

AB We karyotypically analyzed cultured melanocytes from a variety of lesions, including congenital and dysplastic **nevi**, primary melanoma, and metastatic melanoma. The cells derived from congenital **nevi** had normal karyotypes, as did 22 of the 26 cultures derived from dysplastic **nevi**. The karyotypes of melanocytes from primary and metastatic melanomas were all abnormal. The only **chromosome** change in common between the **nevi** with abnormal karyotypes and the melanomas was the **loss** of one copy of **chromosome** 9 (two of four **nevi** and four of 11 melanomas, including three from the same patient) or the **loss** of the short arm of **chromosome** 9, especially of region 9pter-p22 (three of 11 melanomas). We suggest that deletion of a gene or genes on 9p, possibly interferon genes, is an initial step in the malignant transformation of melanocytes.

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     (FILE 'HOME' ENTERED AT 07:17:13 ON 29 OCT 2003)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:17:56 ON 29 OCT 2003
L1          4335 S SPITZ OR BENIGN (3A) SKIN
L2           220 S L1 AND (GAIN OR LOSS)
L3            83 DUP REM L2 (137 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 07:23:28 ON 29 OCT 2003

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:25:46 ON 29 OCT 2003
L4         26902 S NEVI OR NEVUS
L5           915 S L4 AND (GAIN OR LOSS)
L6           261 S L5 AND CHROMOSOME
L7           137 DUP REM L6 (124 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 07:27:36 ON 29 OCT 2003

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:28:43 ON 29 OCT 2003

     FILE 'STNGUIDE' ENTERED AT 07:28:43 ON 29 OCT 2003

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:30:07 ON 29 OCT 2003

     FILE 'STNGUIDE' ENTERED AT 07:30:07 ON 29 OCT 2003

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L7 ANSWER 8 OF 12 MEDLINE on STN
ACCESSION NUMBER: 2000062984 MEDLINE
DOCUMENT NUMBER: 20062984 PubMed ID: 10594753
TITLE: Molecular cytogenetic analysis of **Spitz nevi** shows clear differences to **melanoma**.
AUTHOR: Bastian B C; Wesselmann U; Pinkel D; Leboit P E
CORPORATE SOURCE: Cancer Genetics Program, Cancer Center, University of California San Francisco, 94143-0808, USA.. bastian@cc.ucsf.edu
SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1999 Dec) 113 (6) 1065-9.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000113

AB **Spitz nevus** is a **benign** neoplasm of melanocytes that can be difficult or impossible to distinguish from **melanoma** by clinical and histopathologic examination. We studied genomic DNA from 17 **Spitz nevi** by comparative genomic hybridization (**CGH**). Thirteen lesions showed no chromosomal aberrations, three cases had a **gain** involving the entire p-arm of chromosome **11**, and one case showed a **gain** of chromosome 7q21-qter. Fluorescence in situ hybridization (FISH) on lesional tissue with a probe for the p-arm of chromosome **11** showed 6-**10** p-arm signals per nucleus in those cases with a **CGH**-detected **gain** of chromosome 11p. One case with a normal **CGH** profile also showed increased copy number of 11p by FISH. Thus, the majority of **Spitz nevi** have a normal chromosomal complement at the level of **CGH** resolution; however some may contain **gains**, with 11p apparently being the most frequently involved location. These findings differ significantly from the previously reported changes in primary cutaneous **melanoma**, which show frequent deletions of chromosomes 9p (82%), 10q (63%), 6q (28%), and 8p (**22**%), as well as **gains** of chromosomes **7** (50%), **8** (34%), 6p (28%), 1q (25%) by **CGH** analysis. These clear differences in the location and frequencies of chromosomal aberrations in **Spitz nevi** and primary cutaneous **melanomas** could represent a basis for developing adjunctive techniques for refining accuracy in the difficult differential diagnosis of spitzoid **melanocytic** neoplasms.

L7 ANSWER 3 OF 12 MEDLINE on STN
ACCESSION NUMBER: 2002062077 MEDLINE
DOCUMENT NUMBER: 21645135 PubMed ID: 11787857
TITLE: Spitzoid malignant **melanoma** with lymph-node metastasis. Is a copy-number **loss** on chromosome 6q a marker of malignancy?.
AUTHOR: Mihic-Probst D; Zhao J; Saremaslani P; Baer A; Komminoth P; Heitz P U
CORPORATE SOURCE: Department of Pathology, University Hospital, Zurich, Switzerland.. daniela@mihic@pty.usz.ch
SOURCE: VIRCHOWS ARCHIV, (2001 Dec) 439 (6) 823-6. Journal code: 9423843. ISSN: 0945-6317.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020128
Entered Medline: 20020125

AB Distinction of spitzoid malignant **melanomas** (SMM) from **Spitz nevi** may be difficult or even impossible on the basis of conventional histology. In this report, a patient suffering from a primary lesion diagnosed as a **Spitz nevus** and a metastatic malignant **melanoma** approximately **4** years thereafter is described. A diagnosis of SMM was made subsequently upon review of the primary lesion. In the present analysis, we used comparative genomic hybridization (**CGH**) to define markers characteristic of SMM. The primary lesion revealed deletions on chromosomes 6q and 9p. In the metastasis, additional deletions on chromosomes 10p and 10q and **gains** of chromosome **7** were found. To our knowledge, no chromosomal aberration on chromosome 6 was hitherto demonstrated in **benign melanocytic nevi**. Findings reported in the literature suggest that human **melanoma** metastasis suppressor gene maps to 6q. In contrast, **losses** on chromosome 9p seem to be an early event in the development of **melanoma**. However, they are not only found in **melanomas** but are occasionally present in **Spitz nevi** as well as in atypical **nevi**. The **CGH** result with deletion of 6q in this difficult to diagnose primary **melanocytic** lesion strongly supports the diagnosis of malignant **melanoma**. To demonstrate the reliability of **loss** on chromosome 6q as a marker of SMM, a larger number of lesions must be investigated.